```
  FILE 'BIOSIS, LIFESCI, JAPIO, USPATFULL, EUROPATFULL, CONFSCI, MEDLINE,
    CAPLUS' ENTERED AT 18:00:54 ON 01 JUL 2003
L1            16 S (NELL-1)
L2             2 S (NELL-2)
L3            15 S (NEL-LIKE)
L4             0 S (NEURONAL EPIDERMAL GROWTH FACTOR-LIKE)
L5             8 S (NEURAL THROMBOSPONDIN)
L6        145311 S PROTEIN KINASE C
L7           277 S L6 AND (INTERACTING PROTEINS)
L8           111 S L7 AND (PKC)
L9            85 DUP REM L8 (26 DUPLICATES REMOVED)
L10           11 S L9 AND (EPIDERMAL GROWTH)
L11           11 DUP REM L1 (5 DUPLICATES REMOVED)
L12          129 S MATSUHASHI, S/AU
L13           97 DUP REM L12 (32 DUPLICATES REMOVED)
L14        13844 S 13 AND EGF
L15            2 S L13 AND NELL
L16           48 S TING, KANG/AU
L17           36 DUP REM L16 (12 DUPLICATES REMOVED)
L18           10 S VASTARDIS, HELENI/AU
L19            5 DUP REM L18 (5 DUPLICATES REMOVED)

    FILE 'STNGUIDE' ENTERED AT 18:28:27 ON 01 JUL 2003
L20            0 S MULLIKEN, JOHN B/AU
L21            0 S MULLIKEN, JOHN/AU

    FILE 'BIOSIS, LIFESCI, JAPIO, USPATFULL, EUROPATFULL, CONFSCI, MEDLINE,
    CAPLUS' ENTERED AT 18:32:19 ON 01 JUL 2003
L22            7 S MULLIKEN, JOHN/AU
L23          229 DUP REM L7 (48 DUPLICATES REMOVED)
L24           36 S SOO, CHIA/AU
L25           27 DUP REM L24 (9 DUPLICATES REMOVED)
L26            4 S  TIEU, ANDY/AU
L27            3 S DO, HUY/AU
L28            2 S KWONG, EMILY/AU
L29            4 S BERTOLAMI, CHARLES/AU
L30            0 S KAWAMOTOA, HENRY/AU

    FILE 'BIOSIS, LIFESCI, JAPIO, USPATFULL, EUROPATFULL, CONFSCI, MEDLINE,
    CAPLUS' ENTERED AT 18:40:28 ON 01 JUL 2003
L31            0 S KAWAMOTOA, HENRY/AU
L32            3 S KAWAMOTO, HENRY/AU
L33           73 S KURODA, SHUNICHI/AU
L34           50 DUP REM L33 (23 DUPLICATES REMOVED)
L35           14 S LONGAKER, MICHAEL/AU
L36            4 S L35 AND NELL

    FILE 'STNGUIDE' ENTERED AT 18:47:09 ON 01 JUL 2003

=>
```

36 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AB Previously, we reported **NELL**-1 as a novel molecule overexpressed during premature cranial suture closure in patients with craniosynostosis (CS), one of the most common congenital craniofacial deformities. Here we describe the creation and analysis of transgenic mice overexpressing **Nell**-1. **Nell**-1 transgenic animals exhibited CS-like phenotypes that ranged from simple to compound synostoses. Histologically, the osteogenic fronts of abnormally closing/closed sutures in these animals revealed calvarial overgrowth and overlap along with increased osteoblast differentiation and reduced cell proliferation. Furthermore, anomalies were restricted to calvarial bone, despite generalized, non-tissue-specific overexpression of **Nell**-1. In vitro, **Nell**-1 overexpression accelerated calvarial osteoblast differentiation and mineralization under normal culture conditions. Moreover, **Nell**-1 overexpression in osteoblasts was sufficient to promote alkaline phosphatase expression and micronodule formation. Conversely, downregulation of **Nell**-1 inhibited osteoblast differentiation in vitro. In summary, **Nell**-1 overexpression induced calvarial overgrowth resulting in premature suture closure in a rodent model. **Nell**-1, therefore, has a novel role in CS development, perhaps as part of a complex chain of events resulting in premature suture closure. On a cellular level, **Nell**-1 expression may modulate and be both sufficient and required for osteoblast differentiation.
AN 2002:517921 BIOSIS
DN PREV200200517921
TI Craniosynostosis in transgenic mice overexpressing **Nell**-1.
AU Zhang, Xinli; Kuroda, Shun'ichi; Carpenter, Dale; Nishimura, Ichiro; Soo, Chia; Moats, Rex; Iida, Keisuke; Wisner, Eric; Hu, Fei-Ya; Miao, Steve; Beanes, Steve; Dang, Catherine; Vastardis, Heleni; **Longaker, Michael**; Tanizawa, Katsuyuki; Kanayama, Norihiro; Saito, Naoaki; Ting, Kang (1)
CS (1) Center for the Health Sciences, University of California, Los Angeles, 10833 Le Conte Avenue, 30-113, Los Angeles, CA, 90095: kting@ucla.edu USA
SO Journal of Clinical Investigation, (September, 2002) Vol. 110, No. 6, pp. 861-870. http://www.jci.org/. print.
ISSN: 0021-9738.
DT Article
LA English

L36 ANSWER 2 OF 4 MEDLINE
AB Previously, we reported **NELL**-1 as a novel molecule overexpressed during premature cranial suture closure in patients with craniosynostosis (CS), one of the most common congenital craniofacial deformities. Here we describe the creation and analysis of transgenic mice overexpressing **Nell**-1. **Nell**-1 transgenic animals exhibited CS-like phenotypes that ranged from simple to compound synostoses. Histologically, the osteogenic fronts of abnormally closing/closed sutures in these animals revealed calvarial overgrowth and overlap along with increased osteoblast differentiation and reduced cell proliferation. Furthermore, anomalies were restricted to calvarial bone, despite generalized, non-tissue-specific overexpression of **Nell**-1. In vitro, **Nell**-1 overexpression accelerated calvarial osteoblast differentiation and mineralization under normal culture conditions. Moreover, **Nell**-1 overexpression in osteoblasts was sufficient to promote alkaline phosphatase expression and micronodule formation. Conversely, downregulation of **Nell**-1 inhibited osteoblast differentiation in vitro. In summary, **Nell**-1 overexpression induced calvarial overgrowth resulting in premature suture closure in a rodent model. **Nell**-1, therefore, has a novel role in CS development, perhaps as part of a complex chain of events resulting in premature suture closure. On a cellular level, **Nell**-1 expression may modulate and be both sufficient and required for osteoblast differentiation.

AN 2002485507 MEDLINE
DN 22220328 PubMed ID: 12235118
TI Craniosynostosis in transgenic mice overexpressing **Nell**-1.
CM Erratum in: J Clin Invest 2002 Nov;110(10):1573
AU Zhang Xinli; Kuroda Shun'ichi; Carpenter Dale; Nishimura Ichiro; Soo Chia; Moats Rex; Iida Keisuke; Wisner Eric; Hu Fei-Ya; Miao Steve; Beanes Steve; Dang Catherine; Vastardis Heleni; **Longaker Michael**; Tanizawa Katsuyuki; Kanayama Norihiro; Saito Naoaki; Ting Kang
CS Dental and Craniofacial Research Institute, University of California, Los Angeles, California 90095, USA.
NC K23DE00523 (NIDCR)
SO JOURNAL OF CLINICAL INVESTIGATION, (2002 Sep) 110 (6) 861-70.
Journal code: 7802877. ISSN: 0021-9738.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200210
ED Entered STN: 20020926
Last Updated on STN: 20030108
Entered Medline: 20021023

L36 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2003 ACS
AB Unavailable
AN 2002:897509 CAPLUS
TI Craniosynostosis in transgenic mice overexpressing **nell**-1
AU Zhang, Xinli; Kuroda, Shun'ichi; Carpenter, Dale; Nishimura, Ichiro; Soo, Chia; Moats, Rex; Iida, Keisuke; Wisner, Eric; Hu, Fei-Ya; Miao, Steve; Beanes, Steve; Dang, Catherine; Vastardis, Heleni; **Longaker, Michael**; Tanizawa, Katsuyuki; Kanayama, Norihiro; Saito, Naoaki; Ting, Kang
CS Dental and Craniofacial Research Institute, School of Dentistry, University of California, Los Angeles, Los Angeles, CA, USA
SO Journal of Clinical Investigation (2002), 110(10), 1573
CODEN: JCINAO; ISSN: 0021-9738
PB American Society for Clinical Investigation
DT Journal; Errata
LA English

L36 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2003 ACS
AB Previously, we reported **NELL**-1 as a novel mol. overexpressed during premature cranial suture closure in patients with craniosynostosis (CS), one of the most common congenital craniofacial deformities. Here we describe the creation and anal. of transgenic mice overexpressing **Nell**-1. **Nell**-1 transgenic animals exhibited CS-like phenotypes that ranged from simple to compd. synostoses. Histol., the osteogenic fronts of abnormally closing/closed sutures in these animals revealed calvarial overgrowth and overlap along with increased osteoblast differentiation and reduced cell proliferation. Furthermore, anomalies were restricted to calvarial bone, despite generalized, non-tissue-specific overexpression of **Nell**-1. In vitro, **Nell**-1 overexpression accelerated calvarial osteoblast differentiation and mineralization under normal culture conditions. Moreover, **Nell**-1 overexpression in osteoblasts was sufficient to promote alk. phosphatase expression and micronodule formation. Conversely, downregulation of **Nell**-1 inhibited osteoblast differentiation in vitro. In summary, **Nell**-1 overexpression induced calvarial overgrowth resulting in premature suture closure in a rodent model. **Nell**-1, therefore, has a novel role in CS development, perhaps as part of a complex chain of events resulting in premature suture closure. On a cellular level, **Nell**-1 expression may modulate and be both sufficient and required for osteoblast differentiation.
AN 2002:723279 CAPLUS

DN 138:13109
TI Craniosynostosis in transgenic mice overexpressing **Nell**-1
AU Zhang, Xinli; Kuroda, Shun'ichi; Carpenter, Dale; Nishimura, Ichiro; Soo, Chia; Moats, Rex; Iida, Keisuke; Wisner, Eric; Hu, Fei-Ya; Miao, Steve; Beanes, Steve; Dang, Catherine; Vastardis, Heleni; **Longaker, Michael**; Tanizawa, Katsuyuki; Kanayama, Norihiro; Saito, Naoaki; Ting, Kang
CS Dental and Craniofacial Research Institute, University of California, Los Angeles, CA, 90095, USA
SO Journal of Clinical Investigation (2002), 110(6), 861-870
CODEN: JCINAO; ISSN: 0021-9738
PB American Society for Clinical Investigation
DT Journal
LA English
RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	148.95	620.52

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-13.02	-44.27

FILE 'STNGUIDE' ENTERED AT 18:47:09 ON 01 JUL 2003

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jun 27, 2003 (20030627/UP).

=>

7 ANSWER 27 OF 36 CAPLUS COPYRIGHT 2003 ACS

AB Previously, we reported the isolation and identification of a complementary DNA (cDNA) fragment of NEL-2 gene, the expression of which was upregulated in clin. premature fusing and fused coronal sutures. The purpose of this study was to investigate the distribution and biol. activity of NEL-2 gene in vivo and in vitro. Our data demonstrate for the first time that NEL-2 gene is preferentially expressed in neural and membranous cranial bone, both of which are neural crest cell in origin. Interestingly, NEL-2 is not expressed in endochondral bone. Furthermore, NEL-2 gene expression is upregulated during unilateral coronal suture fusion. Addnl., over-expression of NEL-2 in osteoblast-like cells appear to enhance mineralization. These data suggest that NEL-2 may play an important role in bone induction and cranial suture fusion.

AN 2000:13857 CAPLUS

DN 132:263593

TI NEL-2 gene is associated with bone formation in craniosynostosis

AU **Ting, Kang**; Zhang, Xuguang; Kuroda, Shun'ichi; Mulliken, John B.; Longaker, Michael T.

CS Departments of Surgery and Orthodontics, University of California, Los Angeles, CA, USA

SO Surgical Forum (1998), 49, 602-604
CODEN: SUFOAX; ISSN: 0071-8041

PB American College of Surgeons

DT Journal

LA English

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT